REMARKS

In view of the above amendments and the following remarks, reconsideration of the outstanding office action is respectfully requested.

The rejection of claims 9, 10 and 13 under 35 U.S.C. § 102(b) is respectfully traversed in view of the above amendments.

Claims 9, 10 and 13 have been rejected under 35 U.S.C. § 102(b) as being anticipated by Bourel *et al.* as evidenced by Bruley-Rosset *et al.* The Examiner states that Bourel *et al.* disclose purified autoantibodies isolated from IVIg, wherein said autoantibodies bind DNP-Lysine or IgG. Further, the Examiner alleges that Bourel *et al.* disclose a composition of said antibodies and a pharmaceutically acceptable carrier. The Examiner also alleges that it is an inherent property of said antibodies that they bind to cytokines or IgG (as supported in Bruley-Rosset *et al.*).

Claim 9 has been amended to read "wherein said autoantibodies are highly enriched in ferritin-binding antibodies". Support for this amendment can be found in paragraphs [00054] and [00055], as well as Figure 3 and Table 2 of the instant application.

Applicants wish to point out that, as demonstrated in the Declaration of Dr. Lemieux submitted on October 31, 2007, the antibodies derived from IVIg and purified on DNP, as taught by Bourel *et al.*, do not react with ferritin (see Table 1 and Fig. 1 of the Declaration). The binding of ferritin to antibodies derived from IVIg and purified on DNP is not statistically distinct form the blank control. Therefore, neither Bourel *et al.* nor Bruley-Rosset *et al.* taught autoantibodies highly enriched in ferritin-binding antibodies as indicated in new claim 9.

Additionally, neither Bourel *et al.* nor Bruley-Rosset *et al.* provided evidence that the purified anti-DNP antibodies were able to induce the formation of immune complexes with serum proteins to any significant extent. Although Bruley-Rosset *et al.* provided evidence showing that their anti-DNP fraction was able to bind to certain cytokines (interleukin-1-β, interleukin-2, interferon-γ, tumor necrosis factor-α, interleukin-1 receptor antagonist), it is well-known by those versed to the field that serum concentrations of these cytokines are in the ng/ml range at the most, and that such concentrations would be insufficient to induce the formation of immune complexes with cross-reacting anti-DNP antibodies to a significant degree. Conversely,

the serum ferritin concentrations range between 10 to 300 ng/ml (c.f. Kratz, A., Ferraro, M., Sluss, P. M., and Lewandrowski, K. B. Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Laboratory reference values. N Engl J Med 351 (15): 1548-1563 (2004); and the Notice of Corrections, N. Engl. J. Med 351(23):2461-2461-b (attached as Exhibits A and B), which is between 10 to 100-fold higher than the serum concentration of the cytokines described in Bruley-Rosset *et al.* Thus, anti-ferritin antibodies would be substantially more efficient than anti-cytokine antibodies at forming immune complexes with serum proteins.

In light of the above, the present amendments thus obviate the rejection, and the rejection should therefore be withdrawn.

Claim 10 has been amended. This amendment is supported by the application as filed, especially in paragraphs [0004] (IgG present in IVIg or immune complexes (IC) formed *in vivo* following infusion of IVIg can interact with complement components such as C1q and C3/C4 and thus reduce the amount of these molecules available to induce cell destruction and tissue damage), [00022] (The term "autoimmune and inflammatory disorders" is intended to mean a group of multiple diseases characterized by an autoimmune reaction to the patient cells or tissues which may be accompanied by an inflammatory response due to the activation of the complement. IVIg are used in the treatment of several of these diseases), [00032] (inhibition of phagocytosis and of complement activation), Figure 6 showing that immune complexes bind to complement, and paragraph [00067] (The beneficial effects of IVIg in inflammatory diseases is thought to be dependent of its ability to scavenge complement fragments such as C3b and C4b, thus preventing their deposition in the tissue targeted by the pathogenic process (Ref. 2). The above results are consistent with this mechanism and further indicate that the autoantibodies present in IVIg may be involved in this process by interacting with activated complement components either directly or through the formed autoIC).

New claims 19 to 21 have been added. New claim 19 finds support in the application in Table 2. New claims 20 and 21 find support in paragraphs [00037] and [00063].

In view of all the foregoing, it is submitted that the present application is in condition for allowance and such allowance is earnestly solicited.

Should any fee deficiencies be associated with this submission, the Commissioner is authorized to debit such deficiencies to the Nixon Peabody Deposit Account No. 50-0850. Any overpayments should be credited to said Deposit Account.

Respectfully Submitted,

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